A CASE OF ACUTE FATAL NEURITIS OF INFECTIOUS ORIGIN; WITH POST-MORTEM EXAMINATION.

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The following case is one of generalized neuritis, of inflammatory and disseminated character, involving the nerves to a greater or less extent from their roots to their terminations, and associated with changes in the muscles on the one hand, and with alterations in the central axis on the other, which may have had a slight share in producing the symptoms.

Death occurred from asphyxia on the seventh day; and at the autopsy, besides the signs of neuritis, the lungs were found crowded with small nodular hæmorrhages, and the spleen enlarged.

The patient was under the care of Dr. M. A. Morris, of Charlestown, who kindly asked me to see him in consultation. This I did once only, and that at the beginning of his illness.

The notes which Dr. Morris has kindly placed at my disposal, supplemented by my own, cover most of the essential points. I did not have conveniences for making an electrical examination, and was partly deterred by the agitated, restless state of the patient from investigating certain symptoms as thoroughly as I ought to have done.

At that time, however, while the diagnosis seemed clear, the fatal issue of the case was not anticipated.

The patient was a man twenty-eight years of age, in good health in all respects, and free from constitutional disease of every kind. He was of a highly nervous temperament, and his father and mother are reported as having been also of nervous temperament, but otherwise well. The patient

himself had been formerly laid up with what was called nervous prostration, but at the time of his illness was in his usual health. He did not use liquor to excess.

On Friday evening, November 28th, 1886, he rode from Boston to his home in Charlestown on the front platform of a horse-car, in a heavy rain storm, and got thoroughly drenched. Before morning he awoke with pain in the left shoulder and across the back.

On the following day he complained of a feeling of stiffness in his muscles all over the body; his gait was weak and unsteady, and he felt a general sense of feebleness in all his movements.

The next day he was only with difficulty able to stand or walk. In the evening of this day (the third of his illness), he was first seen by Dr. Morris, who found him complaining of pain, not only in the left shoulder, but also in the anterior muscular mass of both thighs and of numbness in the toes of both feet. He had also noticed that he had had no early-morning erection of the penis since his illness began, which had previously been habitual.

I will here remark that the patient had been excessive in sexual intercourse,during his married life of several years duration, but not so for the period shortly preceding his illness.

There was no pain in the back, nor girdle sensation, nor any weakness of the sphincters; the pupils were normal; his gait was very unsteady, the legs tending to cross one over the other, so that he nearly fell to the floor; the heart was beating regularly; temperature and respiration were normal; heart sounds normal. Dr. Morris ordered 5 grs. of sodium salicylate every two hours, for the relief of the pain.

The next morning he found the patient perspiring freely, and without pain, except in the hip-joint when he rolled over in bed. He was unable to walk, falling forward on the floor when he attempted to do so. The numbness of the feet had increased, but there was no noticeable loss of sensation to ordinary tests; there was pain on pressure over both sciatic nerves and also on deep pressure over both post.

tibials. The pulse was 80, temperature normal, respiration 20.

On the 29th (the fourth day of his illness), the symptoms had still further increased, but were of the same character as before. The pulse was 80, temperature 99° F., respiration 20. The patient was feeling anxious and restless.

On the following day the pulse and temperature were still nearly normal. The patient found it difficult to raise his legs from the bed, but could with some effort draw them up and push them down.

On December 1st (the sixth day of his illness), the pulse was 84, temperature 99.3° F. The patient had been unable to draw the legs up since the previous night; he could not raise his left hand to his head, but could raise the right with moderate effort; the grasp of the left hand was much weaker than that of the right; he could flex both arms; the calves of both legs were tender to deep pressure; there was some strangling on attempting to drink, and slight cough with expectoration of frothy mucous; the patient was talkative and restless.

On December 2d the pulse was 86, temperature 99.2° F. The patient had been delirious the night before and had not slept; he coughed and raised a great deal of frothy mucus; coarse moist rales were heard over both upper fronts; there was complete paralysis of both legs; pain on pressure over both facial nerves; the conjunctivæ were congested; he had a good deal of difficulty in swallowing fluids, but took, on the whole, a good deal of nourishment.

During the afternoon of this day (the seventh of his illness), the paralysis of the upper extremities increased notably; the cough and expectoration and the injection of the conjunctivæ also increased; swallowing became very difficult.

At about seven in the evening, while propped up in bed and taking some nourishment, he suddenly began to cough and strangle, and became insensible. His wife, who was feeding him, thought that a small bit of soft bread soaked in chocolate which he was trying to swallow might have entered his trachea. On Dr. Morris' arrival, forty-five min-

utes later, the pulse was found to be 120, quite strong and regular; respiration barely perceptible, slow and regular; tongue swollen and discolored. There was no evidence that any foreign body had entered the trachea. He died half an hour later, to all appearance from paralysis of the respiratory functions.

I saw the patient on the fourth day of his illness, and obtained essentially the same history as has been given.

The sequence of symptoms was reported to be as follows:

On the night following the exposure, he had pains in thighs on motion, pain in the left shoulder and back, numbness of the toes of both feet.

The next day he found it difficult to raise the left arm at the shoulder, and had weakness in walking. The day following he began to have numbness in the fingers. At the time I saw him all motions were possible, but most of them very feeble. He could still raise the foot about six inches from the bed with the leg extended, but only by the aid of a sudden impulse. The movements of extension at the knee were fairly good. The muscles of the legs were flabby and the patient was unable to stand alone, but from weakness rather than loss of co-ordinating power, and closure of the eyes did not increase the difficulty. Deep pressure in the regions indicated was painful, but there was certainly no marked loss of sensibility to touch or pricking, though only rough tests were used, the patient's restless condition not inviting to more critical investigation.

In view of the paræsthesia, local tenderness, steady increase and wide-spread bilateral distribution of the muscular symptoms, and yet the absence of complete paralysis, the diagnosis of multiple neuritis seemed to be justified, but there was no indication at the time of my visit of paralysis of the heart or lungs.

The autopsy was made on the day after death, December 3d, by Dr. R. H. Fitz, who has kindly given me the following notes:

"Right side of heart distended with liquid blood; both lungs injected and moderately œdematous; punctate ecchy-

moses throughout the lungs in every part; spleen enlarged to nearly three times the normal size, soft, injected; liver and kidneys deeply injected; nothing abnormal in the appearance of the brain or spinal cord.

"Pathological diagnosis; nodular, pulmonary hæmorrhages; acute splenic hyperplasia."

A portion of the anterior crural, the iliolumbar, and vagus nerves, and part of the left axillary plexus, a portion of the diaphragm with filaments of the phrenic, part of the deltoid muscle, and a piece of one lung, were removed for subsequent examination.

It is to be regretted that these nerves and others were not removed in their whole length, especially in view of the fact that in the closely similar case reported by Rosenheim, which came to my notice within a few days after this examination, localized hæmorrhages were here and there found in the course of the nerves.

The appearances, however, in such portions as were removed, leave no doubt as to the general character of the process.

The nerves were examined both fresh, in osmic acid, and after hardening in Müller's fluid. The same pathological appearances, though varying greatly in degree, were found in all; but the best specimens were obtained from the axillary plexus and the anterior crural, and the description will therefore be based mainly upon these, so far as the examination of the hardened specimens is concerned.

The vagus nerve was not examined after hardening. In the fresh state the most marked appearance was a strikingly beaded arrangement of the myeline, due to an accentuation of the markings of Schmidt. I should hesitate to regard this as certainly pathological, were it not that it occurred in connection with further changes.

The myeline was eaten out near the annular constrictions of Ranvier, but this may have been a purely passive, post-mortem change (v. below). Here and there the myeline was swelled and had wholly lost its characteristic markings, and in spots there was an infiltration of cells such as will be described further on.

Of the phrenic, only a few terminal filaments could be examined, and these looked perfectly healthy. Had a more extensive examination been practicable, changes would doubtless have been found, because a certain proportion of the muscular fibres of the diaphragm had wholly lost their transverse striation, and looked lustreless and granular.

The osmic acid appearances in the other nerves were of the usual kind, but the larger number of the fibres examined looked fairly healthy.

Sections of the anterior crural nerve, obtained after hardening and stained with carmine, showed under a low power a streaked or mottled appearance, as if a number of nerve tubes here and there had been blotted out and a new formed substance, taking the carmine stain, had taken their place. When the sections were examined with higher powers, the outlines of nerve fibres were seen more or less altered in the affected parts; yet, nevertheless, the spots seemed of a uniform red color.

The axis cylinders in many of the nerve bundles were to all appearance normal, or nearly so, except in the spots above described; but in some bundles, on the other hand, the same series of striking changes had taken place, which will be described more at length in connection with the axillary plexus.

Here and there were foci of cell formation or infiltration, but on the whole the changes were less marked than in the left axillary plexus.

The lesions met with in the axillary plexus were of the following character:

1. Infiltration of small round cells with granular contents, together with an admixture of cells of other kinds, especially a number having a nucleus of about the size of a leucocyte, and granular contents, but with a distinct body of a pale, homogeneous protoplasm, giving to the whole cell a spherical or oblong shape.

These were by far the most numerous toward the periphery of the nerve bundles, and especially between the sheath and the fibre, and next, around the blood vessels, where they often formed a well-marked ring, spreading thence

outward among the nerve fibres. They were also met with, however, remote from either sheath or vessels, following the course of the nerve fibres themselve; so much so, that I have some preparations of isolated fibres surrounded, at a part of their course, by quite a mass of these cells. Sometimes they were collected into columns which lay between and parallel to the nerve fibres.

I was not able to make out that any of these cells lay actually inside the nerve sheath, except possibly in one or two instances; nor did the nuclei of the nerve fibres appear to be increased in number. Some of the cells were evidently in process of multiplication, the nucleus being divided by a sharp line into two parts. Of these I saw perhaps, three or four well-marked instances.

The degree of this cell-infiltration varied greatly in different sections from the same nerve.

Besides the cells described, there were numerous spindle-shaped cells belonging to the connective tissue, whether increased in number or not, I am not prepared to say, and also here and there larger granular masses which may have been protoplasmic with particles of myeline, or may have been simply altered masses of myeline.

There were also large and highly granular cells, with a large nucleus and irregular, oftentimes flattened border, which I took to be endothelial cells, normal or more or less altered.

I searched for so-called "Mast-zellen" with various aniline colors, as indicated by Rosenheim, but succeeded in finding only one or two that seemed to be characteristic.

2. The next most striking change affected the axis-cylinders, which were in some places greatly enlarged, in others more or less atrophied, in other, again, entirely destroyed.

The distribution of these changes was largely by nerve bundles; that, is, one bundle might show nearly normal axis-cylinders, while in the next bundle they were greatly altered. In some sections there were whole (secondary) nerve bundles, in which scarcely a single axis-cylinder was to be seen; in others all the different changes were repre-

sented, showing that the swelling, atrophy, and disappearance were parts of the same process of destruction.

The changes were also much greater in some strands of the axillary plexus than in others.

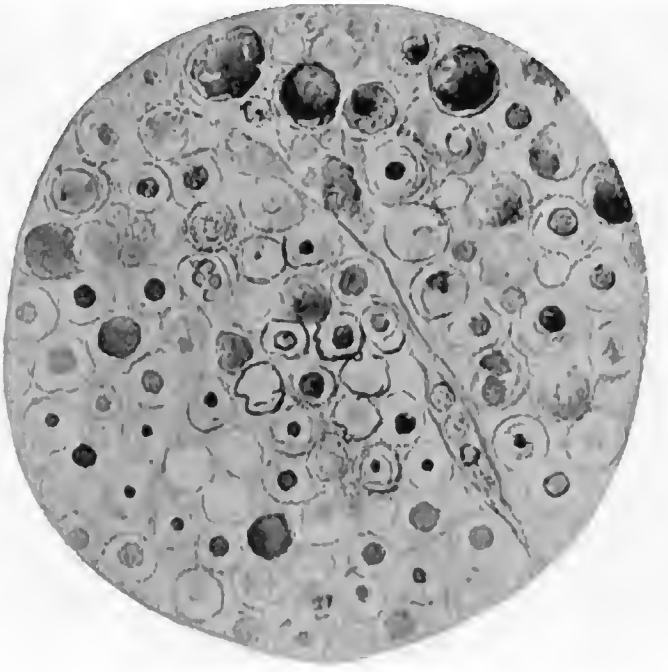

PLATE I.

Section from axillary plexus, illustrating the alteration and destruction of axis-cylinders.

Some of these swelled axis-cylinders occupied the entire thickness of the nerve tube, and even the nerve tube itself seemed sometimes to be distended. In other cases, the sections of the swelled axis-cylinders appeared not round but crescentic, occupying half or two-thirds of a nerve tube.

In order to study more closely the position and character of these changes in the axis-cylinder, I made a series of longitudinal sections, and also of isolated nerve-fibre preparations. Through these it became evident that the swelling occurred here and there in the course of the fibre, and that its most common position was at or near the "annular constrictions" of Ranvier (or "connecting disk" of Schieffer-decker).

In some cases it was obvious what had taken place. The axis-cylinder had become swelled into a bulbous enenlargement, and this had finally burst on one side, leaving half of the shell to give rise to the crescentic sections. In

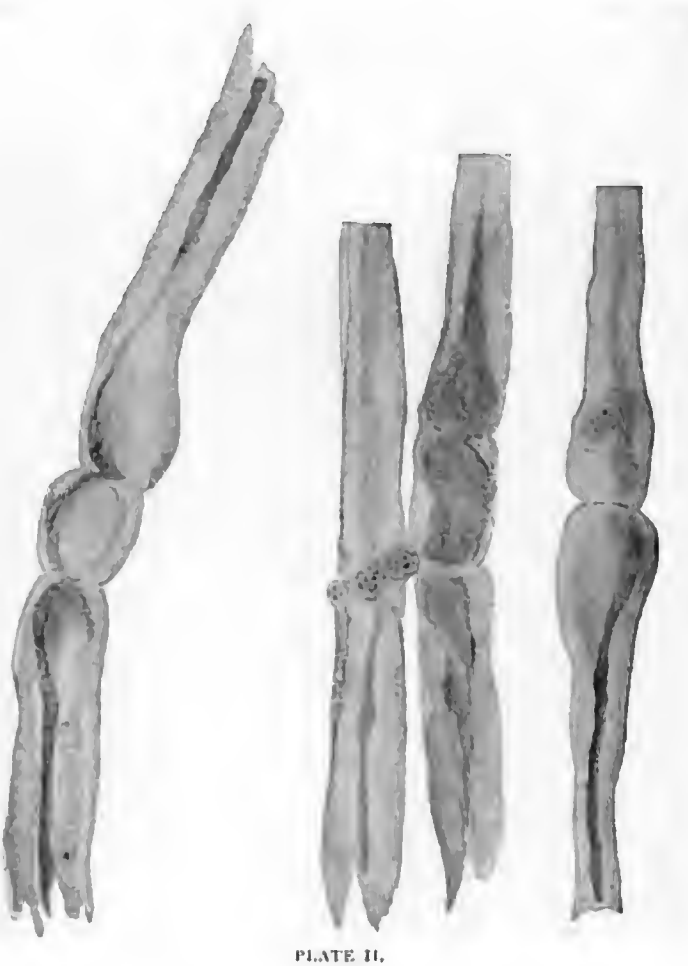

PLATE II.

Nerve fibres with alterations in the axis-cylinders near the constrictions of Ranvier. Hartnack Immersion, one-tenth.

other cases the appearance suggested more or less liquefaction of the altered axis-cylinder (perhaps the myeline as well); for the disk at the annular constriction and the ad-

joining walls of the nerve tube seemed to be, as it were, plastered with a substance coloring strongly with carmine, and evidently formed of the products of degeneration. It is interesting to observe that, as a rule at any rate, the disk had not been broken through, but remained clear and distinct, although, as is shown by the experiments of Boll, Hesse, and Schiefferdecker,[1] on the action of water and other substances upon the fresh nerve, the osmotic or capillary current setting through the nerve fibre is quite competent to break down the connecting disk.

It is worthy of note that these changes of the axis-cylinder, though almost always near the connecting disk, are not always most marked exactly at that point, but often at a little distance back.

The question arises, Were these changes which have just been described wholly or in part post-mortem in character? In one sense I think this question can be confidently answered in the affirmative. That is to say, it is highly probable that the swelling, etc., occurred after the death of the particular nerve fibre; but, on the other hand, it is equally probable that they did not occur in the process of the hardening of the preparation. My reason for that conclusion is, although the same kind of change—that is, swelling and vacuolization—is said sometimes to take place to a certain degree during the hardening of healthy nerves in solutions of chromic salts, yet I have never seen nor read of any change approaching to this in degree, from that cause.

On the other hand, Schiefferdecker describes, as the result of the treatment of fresh nerves with water and dilute acids, a localized swelling of the axis-cylinder, and eventually bursting of the relatively fluid contents through its envelope, which seems to be quite analagous to that which has here taken place.

It is probable that the swelling observed in such cases as this, is of similar origin with that seen in acute inflammation and acute anæmia of the spinal cord.

[1] Arch f. Microsc. Anat., 1887, Bd. xxx., 435.

The character and position of the myeline sheath were also of interest.

In some of the cross-sections, stained with picro-carmine, the nerve tubes could be seen to be still filled with the remains of myeline, even though no axis-cylinders were visible. At times the myeline seemed to have been changed, so that it took up the coloring matter of the carmine to some extent, and it was doubtless in part to this change that the mottled appearance of the cross-sections was due.

Here and there a tube would be entirely empty of myeline for a considerable distance, both in those cases where the axis-cylinders were preserved and in those where they had been destroyed. This may have been partly due to mechanical violence, or to changes during hardening, but it seems hardly possible that it should be entirely accounted for in this way.

The myeline at the annular constriction was, in the bichromate of potash preparations, almost always absent for a certain distance, and this, also, was partly coincident with the alteration in the axis-cylinder at that point, and partly independent of the latter change, occurring in some places where the axis-cylinder ran through the constrictions, as it sometimes did, uninterruptedly and with clear and parallel outlines. So marked was the displacement of myeline at these points that some of the longitudinal sections seemed to be dotted over, under a low power, with vacuole-like spaces.

It will be remembered that Neumann, in his classical paper upon nerve degeneration, points out that the neighborhood of the annular constriction was one of the places where the degenerative changes in the myeline were earliest observed.

Dr. Webber, of Boston, has noted the same fact in some unpublished experiments of his own.

To a certain extent the changes in the myeline at the annular constrictions are probably of post-mortem origin.

It is well known that Ranvier long ago pointed out that when nerves were exposed to the action of water and other fluids for an hour or so after death, the myeline on either

side of the connecting disk would be found rarified and apparently eaten away, and that he considered this change to be an evidence of the fact that nutritive fluids probably enter the nerve at these points.

Schiefferdecker, in the interesting paper above alluded to, while expressing his agreement with Ranvier as to the fact that coloring matters, and probably nutritive fluids, find their way into the axis-cylinders at these points, does not admit that the myeline is dissolved out by these fluids as Ranvier suggested, but considers that it is displaced by the slight mechanical violence in removing the nerve from the body, or of putting it slightly on the stretch, as Ranvier was in the habit of doing as a preliminary to the immersion in osmic acid.

The reason that the displacement of the myeline took place at these particular points is believed by Schiefferdecker to be because the delicate membranous sheath of the nerve, which follows the outline of the fibre and dips down into the narrow portion at the annular constrictions, exerts a lateral pressure where the angle occurs, when it is put upon the stretch.

In other words, the stretched membranous sheath tends to assume the form of a cylinder, the end of which is as large as the connecting disk, but not larger. Consequently, that part of the myeline which occupied the neighborhood of the tapering end of the cylinder is compressed and displaced.

This explanation entirely concurred with the results of some experiments which I had been making, and which will be given elsewhere in detail.

In order to test the point further, I made a number of careful observations upon the nerves of a frog, stretching some of them with a weight of three grammes, and leaving others unstretched.

The results of the experiments were such as to entirely confirm the view taken by Schiefferdecker.

The unstretched nerves, if removed with care, did not show these changes in the relation of the myeline to the connecting disks, either after one hour's immersion in water

or other fluids, or on exposure to the fluids of the body after death for twenty-four hours; whereas the nerves stretched with a weight of three grammes showed the changes very clearly, as Ranvier described them.

Occasionally, even in the unstretched nerve, a fibre is seen in which these changes are observed, but not with sufficient frequence, I think, to invalidate the explanation offered. I believe that there is also reason to think that the putrefactive changes which go on during the twenty-four hours or so after death may make this change occur more rapidly, but this point is still under investigation.

The nerves in the present case were not exposed to any special stretching other than was necessary in their removal, and it is therefore probable that the results were partly the effect of pathological change, and only in part of mechanical violence.

I have, however, seen a similar change, though not nearly to the same extent, in a healthy nerve removed from the body at an autopsy and treated with the same reagents that were used in this case.

One other point should be mentioned in this connection, namely, that the membranous sheath in the neighborhood of the connecting disk, as seen in the hardened specimens, looked as if it had been exposed to pressure from within, making it bulge slightly outward. I have no explanation to offer of the exact manner in which this effect was brought about; but this influence, whatever it may have been (possibly the result of decomposition), may have had its share in the displacement and destruction of the myeline as well.

Changes in the Deltoid Muscles and Diaphragm.—Pieces of the deltoid muscle were examined, both in a fresh state, with and without osmic acid, and after hardening in Müller's fluid. In the fresh specimens the only change observed was that now and then a fibre was seen that had entirely lost its transverse striation, the rest of the fibres being apparently perfectly healthy. The examination of the hardened specimens was more fruitful. The cross-sections showed the size of the fibres to be uniform and normal. There was no trace of the vacuolization or so-called serous atrophy, and

apparently no deposition of fat either within or between the muscular fibres.

The morbid changes were the following:

First, loss of transverse striation, limited in extent, sometimes occupying only a small part of a fibre, the appearance presented being that of fine granulation, sometimes with traces of transverse striation here and there in the midst of the altered substance.

Second, marked infiltration of cells in the connective tissue and around the vessels.

Third, a localized increase in number of the muscle-nuclei, which sometimes, but not always, appeared to be more marked at the place where the transverse striation was wanting.

Fourth, the intra-muscular nerves, as far as could be judged from the few which appeared in the sections, were almost entirely destroyed.

Out of the whole number of fibres making up a small nerve bundle, one or two atrophied axis-cylinders might be seen, as dark, shining points, in picro-carmine sections, the rest of the bundles being represented by altered fragments of myeline with numerous granular round cells lying amongst them.

Fragments of the diaphragm were examined, fresh and with osmic acid, and granular fibres without transverse striation were here and there noted.

Of the central nerve system, the medulla and spinal cord were examined. The brain was preserved, but has not been examined.

In the medulla and spinal cord the following changes were observed: First, in the membranes and in many of the nerve roots, both anterior and posterior, there was an infiltration of round cells both around the vessels and amongst the fibres, and in other respects the nerve roots were more or less changed, the degree of alteration being less than in the peripheral nerves.

This infiltration was rather greater in the lower dorsal region of the cord than in the lumbar or cervical region, or most parts of the medulla.

The blood vessels, both of the membranes and of the central axis, were everywhere crowded with blood. This I take to have been, in part, the result of the asphyxia with which the patient died; in part the sign of an inflammatory process.

Within the substance of the cord the vessels were surrounded here and there with a moderate number of cells contained in the peri-vascular sheath, and the central canal was filled with similar cells.

The nerve cells, so far as I could judge, were essentially normal. Here and there was one with a shrunken or otherwise altered nucleus; but there was nothing, in my opinion, that might not be accounted for by post-mortem changes. The only other pathological appearance in the cord was that here and there at the periphery, especially in the lateral column near the post. cornua, and in the ant. column in the nerve root zone, greatly enlarged axis-cylinders staining feebly with carmine.

The condition of the medulla was carefully examined, in the hope of finding a satisfactory cause for the multiple pulmonary hæmorrhages. There was a general filling of the vessels, large and small, and here and there an accumulation of lymphoid cells in the peri-vascular sheaths, and occasional slight hæmorrhages from the capillaries and smaller vessels.

These disturbances of circulation were, in most of my sections, more marked in and near the sensory nucleus of the vagus and glossopharyngeus than elsewhere, and the hæmorrhages were in fact only seen in this neighborhood. It could not be asserted, however, that the nucleus appeared to have been materially injured.

The vagus nerve roots were affected in varying degrees, and one section, shows a more excessive infiltration than perhaps any other nerve root that I have seen.

It is easy to arrange this case in its proper pathological position up to a certain point. It evidently belongs with such cases as were reported by Eichhorst in Virchow's Arch., vol. 69, 1876, and Rosenheim in the Arch. f. Psych., vol. 18, which the latter has discussed so ably; and

with the other acute, fatal cases of multiple neuritis, none of which, I think, have been of shorter duration than this.

In Rosenheim's and Eichhorst's cases, to be sure, hæmorrhoids existed in the nerves, visible to the naked eye, indicating a more intensely active process than here. On the other hand, nothing could be more intense than the congestion in this case; and the evidences of minute hæmorrhages and cellular infiltration were more extensively present than in most of the other cases, involving the muscles, spinal roots, membranes, and even the central nervous axis to a certain extent, as well as the nerves.

It would be interesting to know whether, if the patient had not died, the spinal changes would have assumed a greater prominence, and a poliomyelitis or a diffuse myelitis have developed itself.

Certainly the topography of the lesions suggests this possibility; but it is to be noted, as regards the question of poliomyelitis, that the posterior nerve roots were quite as much affected as the anterior. The fact that the changes in the white columns (enlargement of axis cylinders) were mainly confined to the periphery, and more marked in the neighborhood of the nerve roots than elsewhere, but they did not seem to occur in the posterior columns. The amount of accumulation of lymphoid cells in the peri-vascular sheaths of the central gray matter was perhaps not great enough to count as the first step in an inflammatory process, though it may have been of that character; but, to say the least, one can hardly doubt that it would have taken little more to precipitate such an event, especially in view of the fact that in the medulla minute hæmorrhages had actually occurred.

The questions of chief importance in connection with this case are: first, as to its etiology, and especially if we can gain through it any light upon the supposed toxic origin of generalized neuritis; next, as to whether these acute, fatal cases exhibit clinical features which will enable us to detect them at their onset.

With regard to the first point, the reasoning, as clearly expressed by Rosenheim, and endorsed by Leyden in his

recent address before the Militär-ärtzliche Gesellschaft of Berlin, is that the great majority of these cases are truly of toxic origin, the source of the poison being the bacteria of tuberculosis or other constitutional disease, or some mineral or other poison. The bacteria are, however, not supposed to themselves be present in the nerve, but only the poisonous substances to which their growth gives rise.

In this way the distinctly infectious cases are brought into a parallel with the cases due to metallic poisoning.

Strümpell, moreover, in a recent paper upon degenerative changes in the spinal cord, although not speaking especially of neuritis, suggests another method in which these processes of poisoning may be started or propagated, namely, through the action of the products of decomposition of the nervous tissues themselves. Dr. Spitzka suggested last year a similar cause for the outbreak of delirium grave.

In Rosenheim's case it was believed that the primary source of the infection was tuberculosis, with which the patient was affected, although at the time of his attack in good nutrition; and he further remarks that, in his opinion, scarcely a case of multiple neuritis has been observed in which no infectious constitutional disease was present. With this view I cannot wholly agree; nor is it maintained, so far as I can see, by Leyden, who himself reports the onset in one of his rapidly fatal cases as being apparently due solely to a condition of exposure and fatigue.

In my case the patient was not the subject of any contagious disease, so far as could be ascertained, and the wetting to which he was exposed was the only cause which could be discovered. Nevertheless, the pathological signs of infection are even stronger in my case than in Rosenheim's and others. In them those signs consisted almost solely in the acute onset and generalized character of the disease, together with the fact that hæmorrhages were present in the nerves. In my case, a marked hyperplassia of the spleen was also present, such as has been observed in several cases of Laudry's disease, but not often and never to this extent, so far as I know, in cases of multiple neu-

ritis. In Rosenheim's case the spleen was reported as measuring twelve centimeters in length, and being soft in consistency.

To what cause to attribute the multiple and nodular hæmorrhages scattered through both lungs, in the present case, I am unable to decide. In view of the absence of hæmorrhages in other regions of the body it would be hardly probable that those were due to the local action of the toxic agent, although this is not to be set aside. It seems more probable that they were of the same origin with the multiple hæmorrhages described originally by Brown-Sequard as due to certain injuries of the medulla, and probably of vaso-motor origin. It is possible that the neuritis of the vagus nerve may have given rise to them, although, so far as I know, such a result has not previously been demonstrated.

It is noteworthy that in this, as in the other important cases of similar kind, the toxic agent, whatever its character, has not given sign of its presence in a diffuse way, but only here and there in foci of limited extent. This is very likely due to the fact that the means of examination at our command do not enable us to detect the first signs of toxic influence. Even if we had any such evidence as that afforded by the examination in Laudry's disease, which seems to be universally considered as most probably of infectious origin, to show that loss of function may precede noticeable alteration in structure, yet we should still, from abundance of facts, be ready to consider that this was possible.

The fact that such a large proportion of the nerve fibres in the affected districts preserved a healthy appearance cannot be taken as a proof that they were performing their functions in a normal manner.

It would not be out of place, before leaving the subject of infection and its possible sources, to refer to the group of symptoms characterized as a new infectious disease and described recently by Weil and others in the Deutches Arch. f. Klin. Med., 1887, 1888.

Taken as a whole, this is not to be confounded for a

moment with neuritis, the high temperature and the jaundice characterizing it as belonging in a different category. It is, however, noticeable that in several of the cases, severe muscular pain and other signs of alteration of the peripheral nervous system were present.

As regards the clinical aspects of the case, I would only mention one or two points.

In the first place, the absence of fever throughout the sickness is noteworthy. This was also noted in Rosenheim's case, and referred to by him as being sometimes present and sometimes absent in the other cases that he describes.

The mode of death seems to have been by respiratory, rather than cardiac paralysis. At any rate, the respiratory symptoms were the striking ones, and although shortly before death the pulse had risen to 120, it is reported by Dr. Morris as having been full and regular, while the respiration was scarcely perceptible.

The distribution of the muscular symptoms deserves a moment's comment. Pierson has advanced the opinion that the paralysis of the cranial nerves is characteristic of the acute cases. This, however, is not endorsed by Rosenheim, who points out, very properly, that these paralyses are seen also in chronic cases, and not necessarily in acute cases only, although when present they indicate a wide generalization of the disease, and may be of bad import. In the present case, the signs of paralysis of the cranial nerves which was noticed, consisted in the difficulty in swallowing and the tenderness over both facial nerves.

The fact that the left shoulder muscles were so early and so severely affected was to me of special interest, because I have been recently collecting cases in order to test the diagnostic value of this distribution of symptoms. There seems to be no doubt that although the extremities are usually the first to be involved, in the multiple neuritis of every cause, yet the larger muscles uniting the limbs and trunk are sometimes affected very early.

Finally, I would call attention to a point which should, perhaps, have guided us to the recognition of the serious

import of the case, which was early presented, namely, the restless, anxious and agitated mental condition of the patient, which seemed at first out of proportion to the severity of his symptoms.

NOTES.—1. Valuable remarks relative to the significance of this acute swelling of the axis-cylinder, may be found in the Arch. f. Psychiatrie, etc., 1887, p. 263, (Auhang), Zur activen Verand, der Axen-cyl., bei Entzundungen, Dr. M. Friedmann; and Bd. XCVI. der Sitzb. der Kais. Akad. der Wissensch. III. Abth. Nov. Heft. 1887. Ueb. die Verand. am Ruckenmark n. Zeitweiser Verschliessung der Banchaorta. J. Singer (Reprint, p. 12).

2. Otto Dees, (Arch. fur Psych., etc., 1888, p. 97), the latest writer on the anatomy and physiology of the vagus nuclei, mentions, incidentally as his opinion that the large "dorsal" nucleus is vaso-motor in function. It cannot, unfortunat ly, be stated whether in the present case the fibres and ganglia of the sympathetic system were healthy or diseased.

DISCUSSION.

DR. WEBBER had four years ago had a large number of cases under observation in the Boston City Hospital, with three deaths. The history of Dr. Putnam's case was similar to that of these fatal cases. The changes about the constriction of Ranvier noted by Dr. Putnam he had found, especially in the earlier stages of the disease in animals, his experiments were upon rabbits and guinea pigs. In one fatal case he had found but little change about the node of Ranvier; he could not say whether there was traction used in removing it or not. Even where the nerves were extensively altered, there would be some fibres intact. The process seemed to affect the coarser fibres rather than the finer. At the time when there had been so many cases in the course of a few months, there had also been prevalent a sickness among horses attended with weakness and temporary paralysis. He had tried to obtain nerves from the horses, but did not succeed.

DR. PYE SMITH, of London, referred to a case of his own, in which both alcoholism and gout were excluded. He inquired in regard to the position of gout in the etiology of the affection here. He understood that gout was rare in the States, yet in speaking with Dr. Welch, of Baltimore, he had learned that the metatarso phalangeal articulation of the great toe often showed evidence of gout on autopsy.

The speaker inquired also in regard to the prevalence of that enigmatical affection called Landry's paralysis. In Landry's original paper there was but little to correspond with the superstructure which had been reared upon it. Landry had stated that there were no post mortem changes. The speaker thought that it must be a very rare condition. It would be especially interesting to know whether cases examined by modern methods of investigation gave equally negative anatomical results.

DR. VAN BIBBER referred to a case of his own, in which the facial nerve had been affected. The attack had passed off, and the patient recovered.

DR. DANA had not met with or found reported a case in which gout had given rise to multiple neuritis. Local neuritis was not uncommon from gout. He had seen one and perhaps two cases of multiple neuritis from rheumatism. In one case there had been for several years attacks of rheumatism each fall. One fall, in place of the usual attack, the patient had an attack of typical multiple neuritis.

DR. PUTNAM inquired whether any gentleman had met with the multiple pulmonary hæmorrhages present in his case.

DR. WEBBER stated that in the fatal case which he had examined there had been no such hæmorrhages, but in this case the vagus had not been affected, while in Dr. Putnam's in was.

DR. PUTNAM said that true gout was rare, at least in Boston. Neuritis did accompany rheumatism, however, both as a local and a general disease.

In regard to Landry's paralysis, there was, he thought, such a group of symptoms not due to multiple neuritis, and not due to spinal trouble.

DR. SEGUIN presented Dr. Shaw's regrets at being absent from the meeting, and exhibited for him three slides, representing a case of locomotor ataxia.